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THE EXTRAOCULAR MUSCLES

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CHAPTER CONTENTS

INTRODUCTION

Think for a moment about how you are reading this text. The fovea in each of your eyes are organized to provide maximal visual acuity, but to make the best use of this you need to continually bring visual targets – here, there, everywhere – into central vision. In order to smoothly or suddenly shift our visual attention, we can:

- rotate our entire body (keeping head and eyes fixed), pretty much like a crab or a fish;
- rotate the entire head, which is what reptiles and birds mostly do; or
- move just our eyes, provided they move “in synch” (conjugate gaze).

Control of eye movements involves a number of central nervous system (CNS) regions and structures in the orbit that we have encountered previously. There is a hierarchy of control systems, including regions within the cerebral cortex, basal ganglia and superior colliculus, which communicate with eye movement centres in the brainstem. These centres in turn directly regulate the output of somatic motor cranial nerves that activate our extraocular muscles. In this chapter, the structure and function of the extraocular muscles will be outlined as well as the higher brain centres involved in the control of eye movement.

SIX MOVEMENTS; SIX MUSCLES

The human eye is very mobile, although it is somewhat tethered by its optic nerve posteriorly. Movement in any direction is a composite relative to three orthogonal axes, and so six basic turning movements can be identified. There are six muscles which directly move the eye, but it is only for two of the six that one muscle corresponds exactly to one action.

Major Movements: For each axis there is a pair of opposite movements. To understand the terminology think of each movement as relative to the nose.

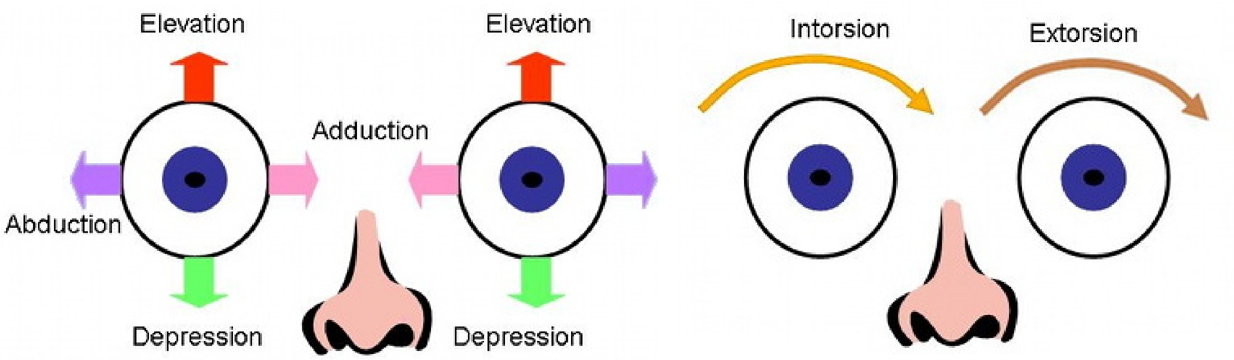

Figure 8.1: *movements of the eyes in six different directions.*

Horizontal movements are: adduction (towards the nose) or abduction (away from the nose); the vertical movements are elevation and depression. Torsional movements are, by convention, intorsion (superior part of eye rotates towards the nose), and extorsion (superior away from nose).

THE SIX EXTRAOCULAR MUSCLES

There are four rectus muscles, arranged evenly around the globe, and all have their origin on the tendinous ring (annulus of Zinn) which is near the apex of the orbit. Each of these therefore pulls in a direction which is about the same as that of the optic nerve. The actions of the two oblique muscles are more complicated and are explained below.

The six extraocular muscles are (Figure 8.2):

- Superior rectus
- Inferior rectus
- Lateral rectus
- Medial rectus
- Superior oblique
- Inferior oblique.

Here, these names are abbreviated as: MR, LR, SR, IR, SO and IO. In a typical eye movement there is simultaneous coordinated action by several muscles (and deliberate relaxation by their antagonists).

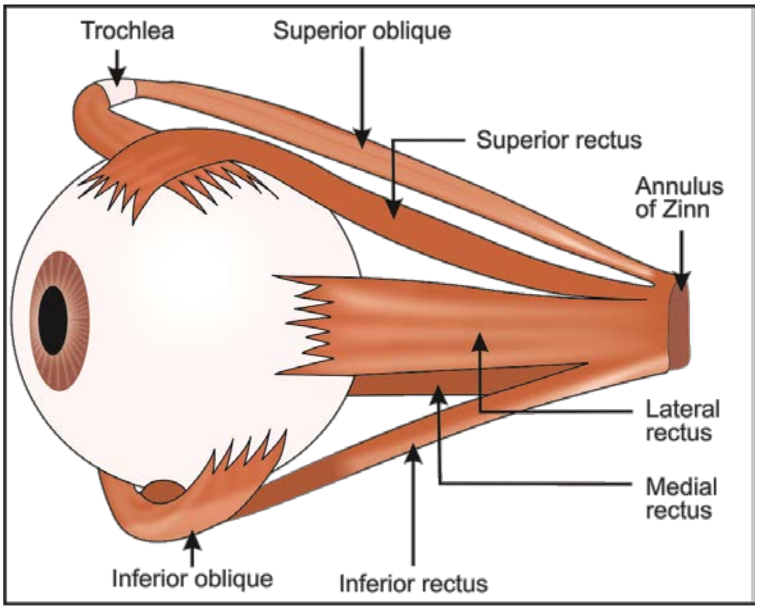

Figure 8.2: The six extraocular muscles causing eye movement (left eye)

THE ACTIONS OF THE EXTRAOCULAR MUSCLES

The keys to understanding eye movements are appreciating the arrangement of the 6 muscles within the orbit, and for each knowing the direction of pull in relation to its insertion into the globe. The key features are:

1) Vertical eye movements are the result of coordination between four muscles. (That is: SR, IR, SO and IO.)
2) Each of these four muscles is considered to have several distinct actions, termed primary, secondary and tertiary actions.
3) The overall action of a muscle often varies depending on the position of the eye, because of varying contributions from non-primary actions.

Innervation to the six extraocular muscles is as follows: the abducens nerve (6^{th} nerve) supplies lateral rectus, the trochlear nerve (4^{th} nerve) supplies superior oblique, and the oculomotor nerve (3^{rd} nerve) supplies the remaining four muscles: SR, MR, IR and IO. It might help to remember this as: LR6, SO4, and 3.

To learn the geometry, think of the eye as a globe of the world, with the cornea at the North Pole. Note how the four rectus muscles all insert at the same “latitude” in the northern hemisphere (See Figure 8.3)

Horizontal eye movements are controlled entirely by the medial and lateral rectus muscles. Medial Rectus adducts the eye (movement to the midline), while Lateral Rectus abducts the eye.

The LR passes anteriorly from the Annulus of Zinn to pierce Tenon’s capsule and insert 6.9mm behind the limbus. The lateral check ligament limits action. Innervation is by VI (abducens) cranial nerve.

The MR is attached to the dural sheath of optic nerve at Annulus of Zinn. It passes through Tenon's capsule and inserts 5.5mm behind limbus. The medial check ligament limits its action. Innervation is by inferior division of the cranial nerve III.

For vertical eye movements, the Superior Rectus and Inferior Oblique combine to cause elevation; while Inferior Rectus and Superior Oblique combine to cause depression.These rectus actions will first be studied in more detail, and then the reason why the obliques act differently will be explained (e.g. why SR elevates but SO depresses).

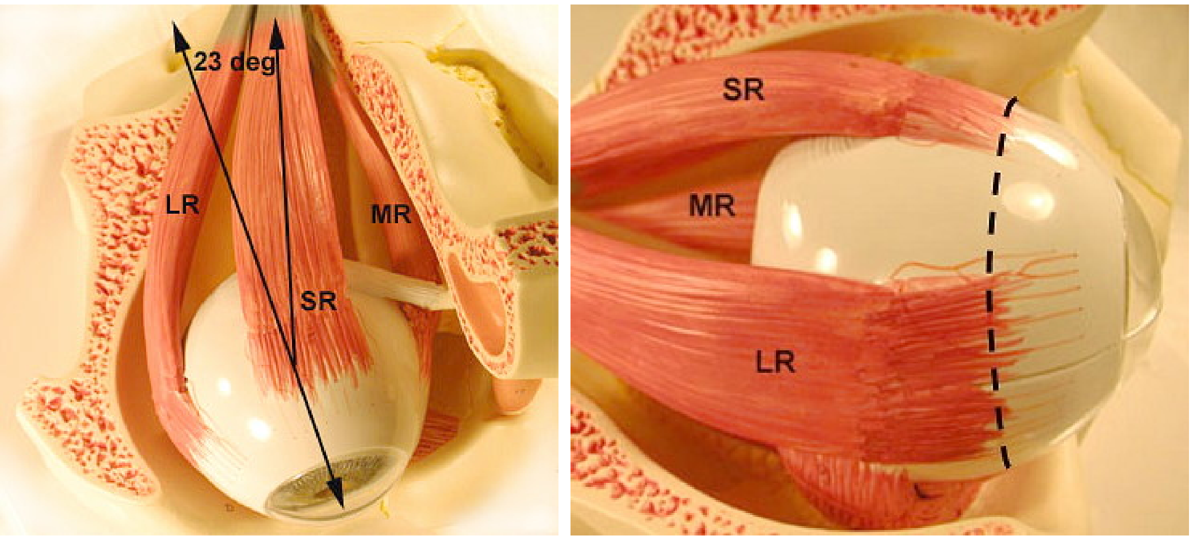

Figure 8.3:*Anatomy and insertion of extraocular muscles*

Obviously, the Superior Rectus inserts into the superior part of the eye. It is the longest of the rectus muscles, and is attached to the dural sheath of the optic nerve at the Annulus of Zinn. It passes forwards and laterally to pierce Tenon's Capsule and inserts into the sclera 7.7 mm posterior to the corneo-scleral junction (*limbus).* It is connected to the levator muscle by a band of tissue between the Tenon's Capsule sheath. The SR is thus synergistic in action with the levator. Innervation is by the superior division of the cranial nerve III (oculomotor).

The body of the muscle is at an angle of 23° medial from the visual axis (primary gaze position). When this muscle contracts, the primary action is to elevate the eye. The slightly medial pull (on its anterior insertion) means it will also adduct (turns eye towards the nose), and intorts the eye.

Inferior Rectus (not shown in 8.3) is very similar except that it inserts into the inferior part of the eye. Thus it primarily depresses the eye, and also, but with less strength, adducts and extorts the eye. From the Annulus of Zinn, the IR passes anteriorly and laterally, pierces Tenon's capsule and inserts into the sclera 6.5mm behind limbus. Innervation is provided by the inferior division of the oculomotor (III^{rd}) nerve

Note that for both SR and IR, if the eye is first abducted (by LR) then the 23° angle is abolished, which means the primary actions are maximized and the others become negligible.

The obliques act differently because (a) they both insert posterior to the equator, and (b) they both pull towards the medial front corner of the orbit.

Superior Oblique has its origin posteriorly on the same tendinous ring as the recti. Its muscle belly passes alongside that of MR but its tendon first passes through the trochlea, a ligamentous pulley located on the upper medial wall of the orbit (frontal bone). The SO tendon then passes back underneath the superior rectus to insert behind the SR insertion and behind the equator at an angle of 54 degrees from primary position of gaze. This remarkable anatomy means that when SO contracts the eye is pulled towards the trochlea (instead of towards the muscle's origin). Thus, with the eye in the straight-ahead position, the primary action of SO is intorsion.

In addition, as SO pulls on the posterior part of the eyeball it turns the eye downwards. Therefore Superior Oblique is a depressor of the eyeball. This secondary action is strongest when the globe is an adducted position.

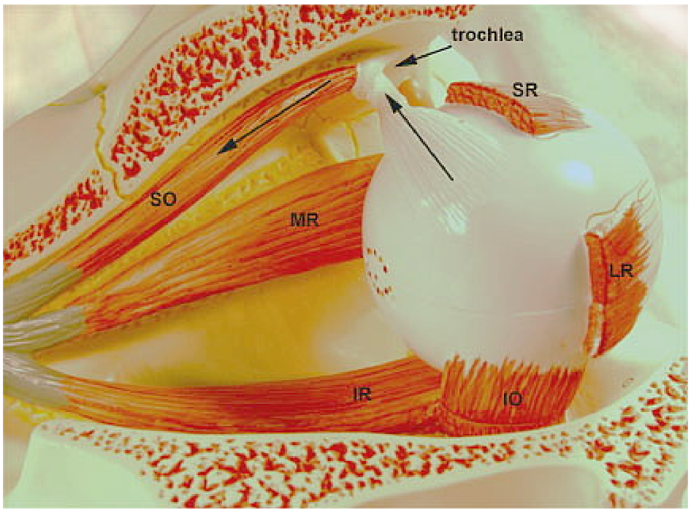

Figure 8.4: *The eyeball showing the position of superior oblique.*

Inferior Oblique has a similar trajectory of pull but acts on an inferior region of the "southern hemisphere". Its origin is on the floor of the orbit near the medial anterior corner (so no pulley is involved, and it is the only one that does not originate from the annulus of Zinn). With IO, the medial pull causes the primary action of extorsion, and the secondary action of elevation is also significant.

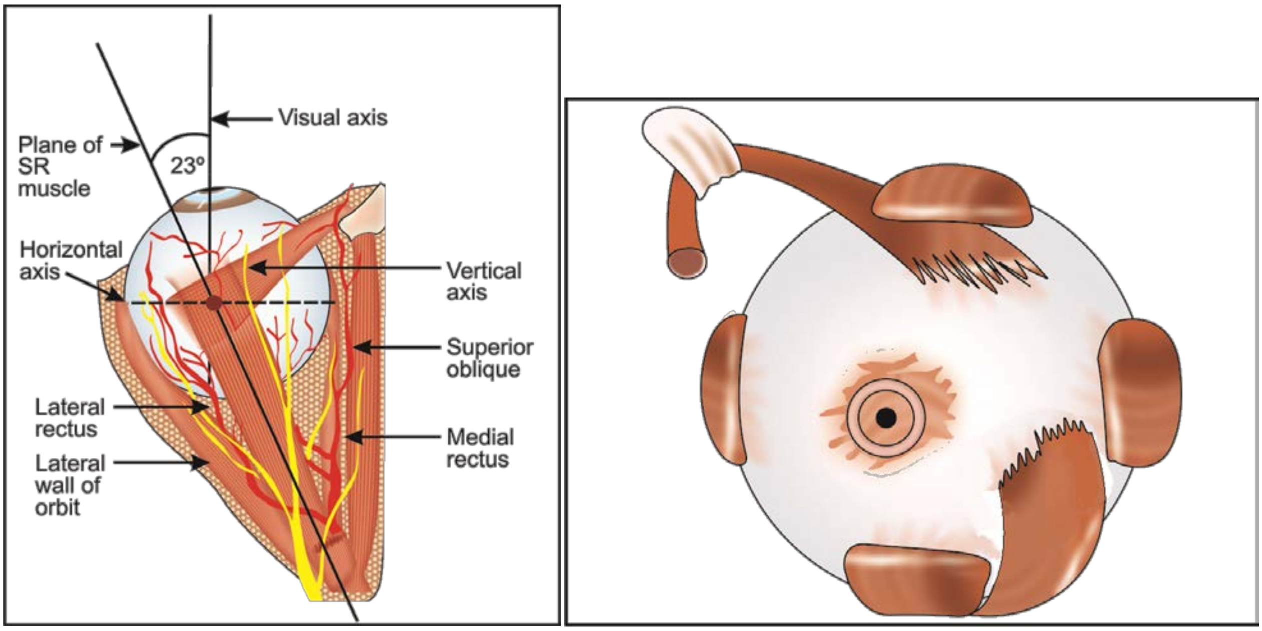

Figure 8.5: *Extraocular muscles, including insertion of superior oblique (R)*

HISTOLOGICAL STRUCTURE OF THE EXTRAOCULAR MUSCLES

The extraocular muscles have microscopic structure qualitatively similar to other striate muscle (Figure 8.6), containing longitudinal fibres, oval in cross section with peripheral nuclei.

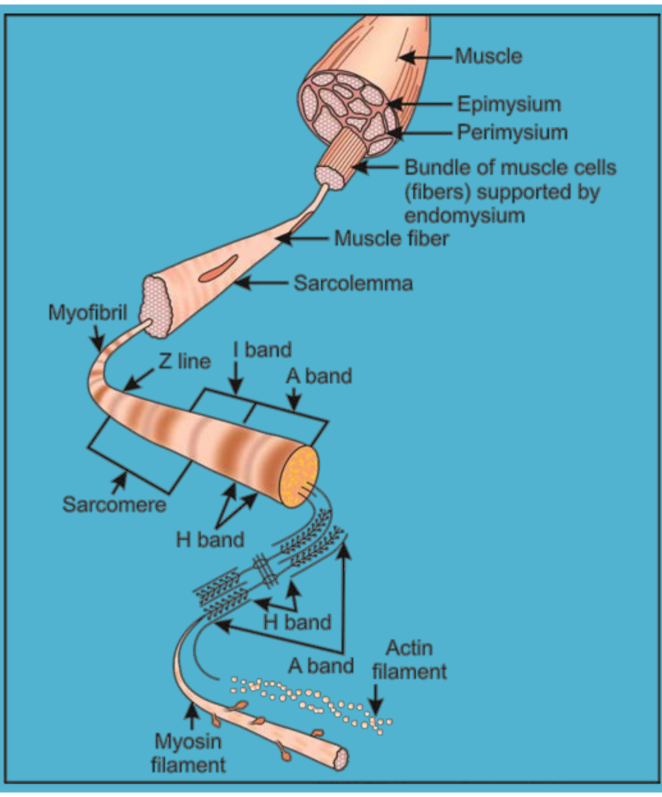

Figure 8.6: *Structure of extraocular muscle (inspired by Snell and Lemp)*

Fibres are bound together by a mixture of collagen and elastic fibres making up the *endomysium*. Continuous with this is the *Perimysium* which surrounds individual bundles of fibres within muscle. An *epimysium* sheath surrounds the outer muscle.

Extraocular (EO) muscle fibres tend to be largest in centre of muscle and smallest at periphery. In contrast to other skeletal muscles, the EO muscles contain delicate connective tissue with many nerve fibres and elastic tissue. They are also more vascular than other skeletal muscle. Each fibre has an outer plasma membrane known as the sarcolemma where the numerous nuclei can be observed, and the inner cytoplasm (specifically known as the *sarcoplasm*) contains the cylindrical cross-section myofibrils. The myofibrils possess repeating *sarcomere* sections which in turn contain *actin* and *myosin* filaments.

Motor innervation is via neuromuscular spindles connecting to either motor end-plates (large and myelinated) or ‘grape-like’ endings (smaller and often unmyelinated). The arrival of a nerve action potential causes a release of acetylcholine from the axon terminal, which in turn causes a depolarization from the resting potential in the muscle fibres through a leakage of both potassium and calcium. This resultant muscle action potential propagates along the nerve fibres and stimulates the release of calcium from the sarcoplasmic reticulum. Upon the binding of calcium, contraction is caused by the sliding of the actin and myosin filaments across each other in a ratchet-like fashion.

The muscle tendons are formed by parallel bundles of collagen and elastic fibres. Collagen fibres enter the sclera and fuse with the collagen within it, thus forming a very tight connection. Elastic fibres terminate abruptly on entry to the sclera.

NEURAL CONTROL OF EYE MOVEMENTS

The human retina has a very small region, the fovea, where visual acuity is at a maximum. The goal when looking at any object is to image that thing at the centre of the fovea. The visual system has a number of ways of achieving this to match various requirements.

i) Reflex Mechanisms

Vestibulo-ocular movements: These compensatory eye movements stabilise the visual image in response to small head movements. Consider what would happen if your eyes were fixed in place, and your head moved. Any image being viewed would be swept across the retina by an amount equal to the head movement – it's the same problem as camera-shake. To avoid this, we have a kind of “Steady-Cam”: each head movement is matched by an eye movement in the opposite direction (and of the same velocity) so that the retinal image effectively remains stationary.

This vestibulo-ocular reflex (VOR) is initiated by the vestibular system within the inner ear (kinetic part of vestibular apparatus). It happens almost every time you move, as there are countless minor head movements you are not aware of. If your head rotates a little to the right the VOR will move your eyes the same amount towards the left. If you tilt your head a few degrees towards your right shoulder, this will be counteracted by torsional eye movements. The circuitry for this also involves the cerebellum and cranial nerve nuclei. However this low-level reflex is quite primitive. Using signals from the inner ear to minimize the motion of retinal images is indirect, and not very precise.

A better strategy is to use visual stimuli to drive correctional eye movements.

Optokinetic reflex: These eye movements occur in response to a large region of the retinal image moving. An example of this reflex occurring repetitively is when one sits in a train and looks out the window at nothing in particular. As the scenery passes by, the eyes briefly follow (fixate on) a scene and then rapidly jump to the next one.

ii) Attention-directed Movements

Saccades: These are rapid ballistic movements that abruptly change the direction of gaze. They are important for placing a different object of interest onto the fovea. In a saccade the rotational velocity of the eyes can be up to 700° per second, for example to suddenly switch from looking leftwards to rightwards. Because the velocity is so high, the movements are very brief, rarely taking more than 50ms from start to finish.

Saccades are voluntary eye movements in the sense that we can choose to look at something or not, and we purposefully shift visual attention. Once initiated, however, execution of the movement is automatic and pre-determined. Visual perception is transiently suppressed during saccadic eye movements, so that we do not see a rapid sweep during the movement. After a saccade has “caught” the target, other smaller eye adjustments hold it.

Here are two examples of visual tasks with typical eye movement trajectories overlaid. No doubt you have been doing such things all the time without realizing how frequent and precisely controlled your eye movements were.

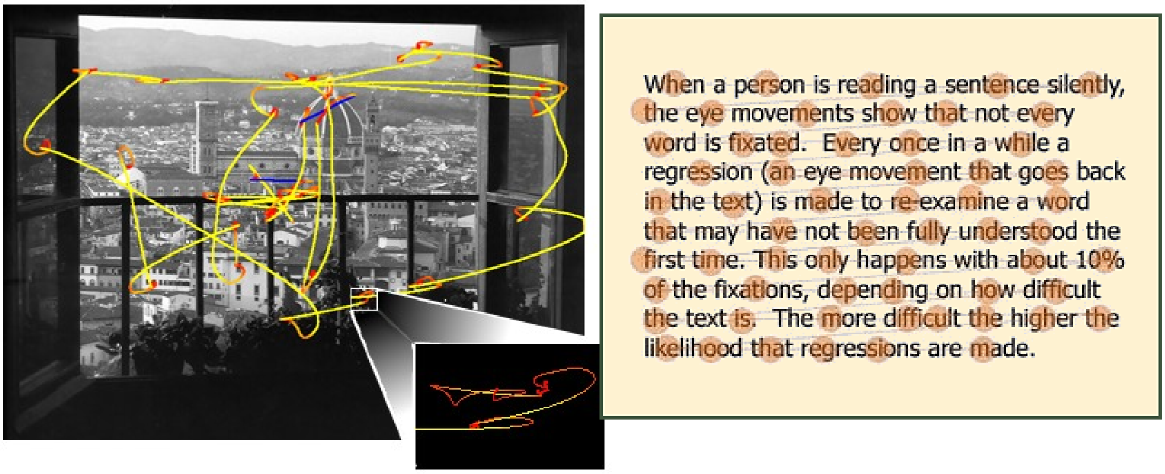

Figure 8.7: *two examples of visual tasks with typical eye movement trajectories overlaid.*

Smooth pursuits: These movements are much slower and are important for keeping a moving target within central vision, in other words visual tracking. They are not under voluntary control.

Vergence movements maintain fixation on an object when it moves closer or further away from you. For example, a ball is thrown to you from 25 metres away and you watch it in order to catch it.

NEURAL CONTROL PATHWAYS

Contraction of other skeletal muscles in the body involves coordinated activity of upper and lower motor neurons. Similarly, there is a hierarchy of control mechanisms important for regulating eye movements. In order for the two eyes to move together in a coordinated fashion, it is too simplistic to think about the eye movements as just what happens at the extraocular muscle level. Indeed, several brain regions act to control eye movements, including the cerebral cortex, the brainstem, the superior colliculus, and cerebellum.

Let's consider first how we move both our eyes laterally in a coordinated fashion. As explained earlier, to move our eyes horizontally requires coordinated actions of the lateral and medial rectus muscles. Consider the diagram below: moving the eyes to the left requires contraction of the left lateral rectus, whilst at the same time relaxation of the left medial rectus. The right medial rectus must contract in unison, and the right lateral rectus must relax.

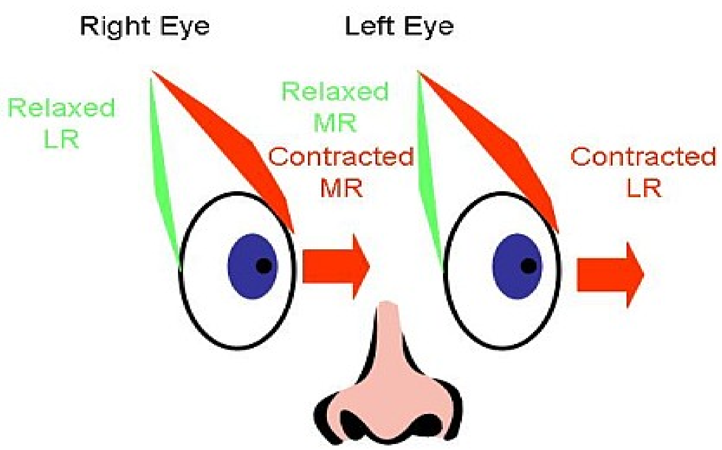

Figure 8.8: *Coordination of eye movements*

The coordination of contraction and relaxation of opposing muscles is mediated by circuits within the brainstem. It involves the cranial nerve nuclei, vertically arranged tracts connecting the cranial nerve nuclei, and the reticular formation. The neural circuit important for controlling horizontal eye movements involves information flow between the abducens (VI) and the oculomotor (III) nuclei. The medial longitudinal fasciculus (MLF) is the tract that links the abducens nucleus on one side with the oculomotor nucleus on the other side. In particular the MLF coordinates contraction of the left lateral rectus with contraction of the right medial rectus (or vice versa). In order to achieve relaxation of the opposing muscles, the paramedian pontine reticular formation (PPRF) is important. This region of the reticular formation provides inhibitory input into the abducens nucleus on the opposite side.

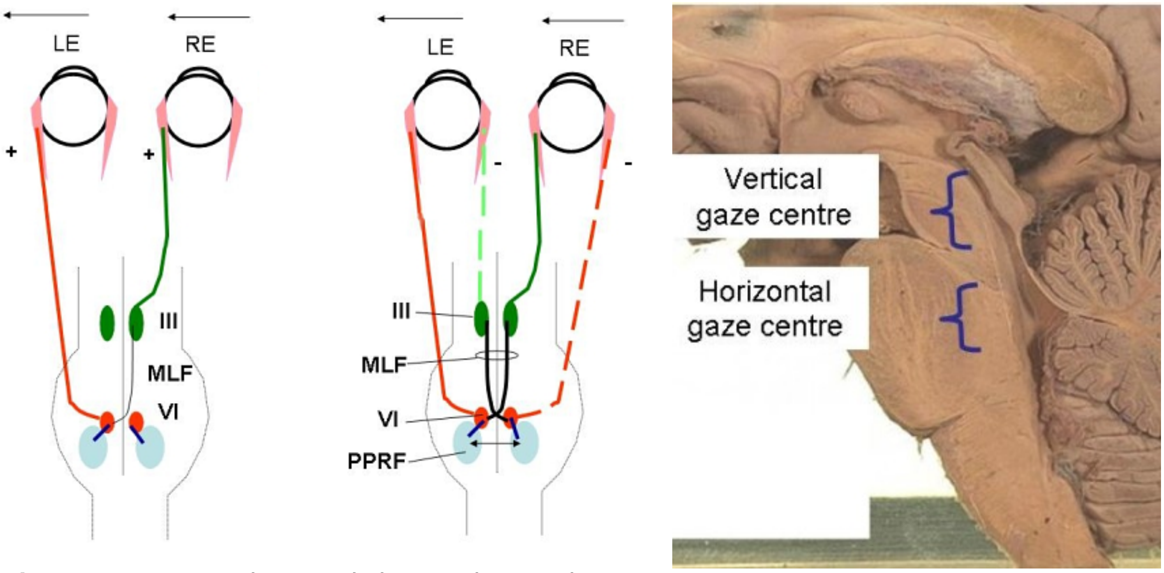

***Figure 8.9:** Extraocular muscle innervations and gaze centres*

As noted earlier, vertical eye movements involve two rectus muscles and two oblique muscles. The brainstem neural circuits that regulate these four muscles in a coordinated fashion are all located within the midbrain and are less well understood. The brainstem centres controlling vertical eye movements are located within the midbrain reticular formation.

Particular clinical syndromes are associated with focal lesions in the brainstem. For example, lesions that affect the pontine gaze centres will lead to deficits in horizontal gaze towards the ipsilateral side, but leave vertical eye movements intact. Conversely, damage to the midbrain can lead to abnormalities of vertical gaze, leaving horizontal gaze intact. If a focal lesion (e.g. multiple sclerosis) affects the MLF on one side, there is typically failure of ipsilateral adduction when the other eye abducts, resulting in diplopia. This syndrome is called internuclear ophthalmoplegia (there are several variants).

Control of saccades also involves complex neural circuitry linking a number of higher brain centres, including parts of the basal ganglia. The expression of this processing is through the brainstem which is therefore a key centre. Patients with lesions in the relevant brainstem region cannot make horizontal saccadic eye movements to the side of the lesion. As noted above, coordination of horizontal eye movements involves the paramedian pontine reticular formation, and it is neurons within this zone that orchestrate the pulse-step output to extraocular muscles.

Two cell types are particularly important for this. Burst neurons in the PPRF fire at a high frequency just before and during ipsilateral saccades (similar to the pulse component of the lower motor neuron). Omnipause neurons fire steadily at all times except during a saccade. These neurons are GABAergic and inhibit burst neurons. There are also a number of different types of burst neurons – such as long-lead burst, medium-lead burst, inhibitory burst, and tonic neurons – that have various roles in modulating brainstem output.

The decisions on where and when to make purposeful eye movements are specified in certain regions of the cerebral cortex. The frontal eye fields, supplementary eye fields and posterior parietal cortex contain neurons that drive eye movements. These brain regions communicate with the superior colliculi which integrate the information and send signals to the long-lead burst neurons in the gaze centres.

Unlike saccades, smooth pursuit and vergence movements involve the cerebellum. This is probably necessary because these eye movements are more likely to be incorporated into active adjustments of head and/or body position, and often are related to dextrous limb movements.

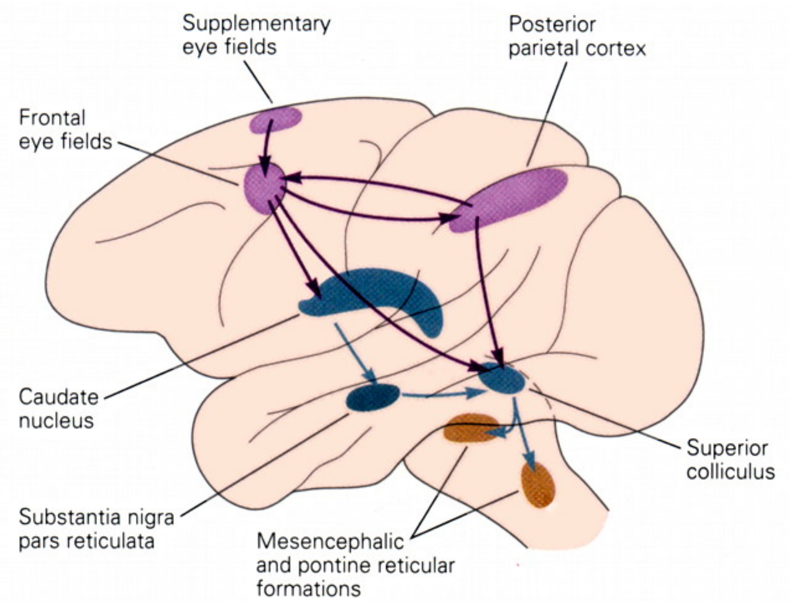

Figure 8.10: *Centres involved in determining adaptive eye movements, depicted in a lateral view of monkey brain.*

EXAMINING EYE MOVEMENTS

If the eyes do not move in a conjugate fashion, a patient will very likely experience double vision (diplopia). This may be caused by trauma to the orbit, an extraocular muscle problem, a nerve defect, or a lesion in the brainstem.

Thus the main aim of testing eye movements is to check for diplopia and to see if the action of each of the six muscles is normal. This in turn provides information about the relevant cranial nerves and regions of the midbrain and pons.

For more information on how to test eye movements, please refer to the chapter *Motilities* in the module *Clinical Optometric Procedures 1*.